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wholly wanting in the flora of the latter region. From these climatic facts it appears that while soil moisture is a condition *sine qua non* of the presence of the cacti, the range of the actual amount of soil moisture must be very great indeed, so, in short, it results that the temperature is the factor in direct control, thus a very important limiting factor.

Should we sum up, therefore, the factors thus far mentioned as being important among those which determine the distribution of the cacti, we find, in the first place, that the shallowly placed root-system subjects the roots to the greatest possible extremes in soil temperatures, including those that are high, and, at the same time, makes it possible for the plants to advantage from the minimum effective rainfall. Further, an effective growth rate of the roots takes place only at relatively high soil temperatures. And, finally, a certain but highly variable amount of moisture must be present in the soil. Since the *crux* of the matter, however, appears to be the fact that the root-system of the cacti are essentially superficial, there is the additional factor, or factors, which bring about this circumstance. These are at present unproved, but the results of experimental studies, not published, indicate that among them must be included the response to the oxygen supply of the soil.

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THE INHERITANCE OF CONGENITAL CATARACT

In the February number of the American Naturalist there is an article from the Bussey Institution by Jones and Mason[1] in which an attempt is made to show that congenital cataract behaves in heredity as a simple Mendelian recessive. The authors from a study of family histories published by Harman in the "Treasury of Human Inheritance" come to conclusions at variance with those of Bateson and Davenport, which authors they are perhaps unjustly disposed to criticize. The paper is well written and embodies a considerable mass of data, so that the reader not familiar with this particular problem might easily be led to think that the older investigators had really made a mistake in interpretation. The evidence, however, does not seem to warrant such a conclusion, as the present paper will attempt to demonstrate.

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[1] Jones, D. F., and Mason, S. L., "Inheritance of Congenital Cataract," The American Naturalist, Vol. L, No. 590, pp. 119–126, February, 1916.

It is stated on page 120 of the article in question that the data used in the paper are taken from the tables accompanying Harman's publication. Since we are concerned wholly with a question of interpretation we may confine ourselves to these tables.[2] The families recorded in the tables are classified by Jones and Mason as follows (p. 120):

> After discarding all the doubtful cases, and picking a sibship with its parents from the table as a family, there is left a total of one hundred and twenty-five families which are classified into three different categories, as follows: (*A*) Both parents normal with at least one abnormal child; (*B*) one parent normal, the other affected with some form of congenital cataract, with at least one abnormal child; (*C*) both parents abnormal, giving only abnormal children.

In each of these groups (*A, B, and C*) it is thought that evidence is found in support of their contention that congenital cataract is a recessive character. We may now consider this evidence in the order in which it is presented.

In group *A*, 31 families are cited in which both parents are normal with one or more affected children. This is the strongest, or really the *only*, evidence that is offered in favor of the recessive character view. Let us examine it more closely. On going over Harman's tables, we find that of these 31 families there are 16 in which the affected individuals produced no offspring or nothing but normals. We do not wish to lay great emphasis on this point, but in such cases one should bear in mind the common clinical belief and the experimental proof (for rabbits and guinea-pigs)[3] that a certain number of congenital cataracts are produced by intrauterine poisoning without necessarily any reference to heredity.

Another possible explanation for some of the examples in group *A* is that they represent cases of origin *de novo*. Jones and Mason say:

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[2] Harman, N. Bishop, "Congenital Cataract," in the "Treasury of Human Inheritance," Part IV, Section XIII *a*. Eugenics Laboratory Memoirs, XI, pp. 126–169. Pl. XXVIII–XXXIII. London, 1910.

[3] Pagenstecher, H. E. "Uber eine Methode der gemeinsamen experimentellen Erzeugung von Augenmissbildungen und von angeborenen Staren bei Wirbeltieren," *Münch. Med. Woch.*, 58 Jahrg., No. 32, pp. 1716–1717. Aug. 8, 1911. (Reviewed in most of the eye journals.)

Surely it is not possible to explain so many cases as *origin de novo* or as due to faulty classification of the parents.

With reference to the origin *de novo* of characters it may be recalled that one does not have to search the literature long to find instances of the same mutation occurring repeatedly in different stocks and at different times, or of certain stocks that seem to be especially prone to mutation.[4] Congenital cataracts occur in many races of man and in other mammals. So far as the writer is aware we are not at present in a position to state, either on the basis of observed data or from *à priori* consideration just how frequently mutations may occur in the human germplasm.

Again, since Jones and Mason elsewhere in the same paper (p. 124) use the argument that "heterozygous individuals sometimes show the recessive character," we might, if necessary, use the same argument to prove the dominance of cataract. On the assumption that congenital cataract is dominant instead of recessive it might be maintained that in those cases where both parents of affected individuals seem to be normal, one of them is, after all, heterozygous, and affected children are therefore to be expected.

Finally it should be recalled that in their statistical study of these 31 families Jones and Mason do not get the results that their hypothesis demands. After having made the proper mathematical corrections there still remains a discrepancy which they do not adequately explain, the agreement between theoretical and observed results being only .418 (p. 122). In order to test what one should expect from the examination of such data when the character is recessive, I have taken a paper by Usher[5] on retinitis pigmentosa and summarized the charts in the same way that Jones and Mason summarize those of Harman. Now retinitis pigmentosa probably *is* a recessive character as is commonly believed. In the charts of Usher are recorded 44 families in which neither parent of the affected individual shows the defect (*i. e.*, both are presumably heterozygous). These 44 pairs of parents are recorded as having 320 children, of whom 77 are affected—24 + per cent. as compared with an expectation of 25 per cent. If the data on cataract were to yield results as close as this we would be more disposed to credit the view that the character is recessive.

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[4] To cite a single case, we may mention the results of Barfurth in breeding fowls. In normally 4-toed races polydactylism occasionally arises *de novo,* but once having appeared is transmitted as a Mendelian dominant. Barfurth, Dietrich, "Experimentelle Untersuchung über die Vererbung der Hyperdactylie bei Hühnern. V. Mittelung: Weitere Ergebnisse und Versuch ihrer Deutung nach den Mendelschen Regeln," *Arch. f. Entwichlungsmech. d. Organism.,* Bd. 40, pp. 279–309, 1914.

[5] Usher, C. H., "On the Inheritance of Retinitis Pigmentosa with Notes of Cases," The Royal London Ophthalmic Hospital Reports, Vol. 19, pp. 130–236. 1914.

So far as the 31 families of category *A* are concerned it must be admitted that absolute proof of the fallacy of the recessive character view can not be furnished, but it will be apparent that there is considerable evidence which not only fails to support this view, but actually points decidedly against it. This fact, taken in connection with the positive refutation which the data in categories *B* and *C* supply, makes a very strong case against the view that congenital cataract is a recessive character.

In the second category (*B*) where one parent is affected the other normal, Jones and Mason remark that "the number of affected children would be expected to be approximately the same whether the character was inherited as a dominant or a recessive" (p. 121). But it must be borne in mind that the offspring of a recessive show the 1 : 1 ratio *only* when the mate is heterozygous, and in their second table Jones and Mason assume that the parents of the children in group *B* represent the cross "Nn × nn." The question is not raised as to the probability of the occurrence of such matings nor does there seem to have been an attempt made to trace the offspring from the normal and affected members of the F_1 generation. In other words, the data of really critical significance do not seem to have been considered. As it stands, then, Table II seems to present no evidence either for or against the above hypothesis, a point which the authors themselves recognize as the quotation indicates.

Since the authors have not tabulated the data which would seem to be of most significance, we may return to Harman's original charts assuming for purposes of the discussion that congenital cataract really is a recessive. On this assumption there are two important conditions which we should expect to find fulfilled.

1. If congenital cataract were a recessive, a cataractous person married to a normal should in most cases produce only normal children. This will be apparent when it is recalled that con-

genital cataract is so rare (perhaps 1 in 4,000 or 5,000)[6] that the number of heterozygous individuals in the general population must be relatively low—theoretically not more than 1:30.[7] In other words, if congenital cataract were recessive the chances that an affected individual in marrying would get a heterozygous partner and thereby be able to produce affected children would be only one in thirty and the chances that the same thing would happen in several generations in direct descent *as occurs repeatedly in the charts* (the case in over 40 *different family trees*) become extremely remote. We should not then expect families with one cataractous parent to contain affected children more often than in the above proportion.[8]

2. If congenital cataract were recessive the normal children of a cataractous parent should themselves produce affected children in half as many cases as do their cataractous sibs and the total number of affected children produced should be one half as great in the first case as in the second. This expectation follows from the assumption that the original (grandparental) mating was Nn × nn. As a result of such a mating the F_1 generation can be composed only of Nn and nn individuals. Neither of these should produce affected children except when married to an Nn (or an nn), and the chances of such a marriage are as great (or as remote) in the one case as in the other. In other words, an equal number of heterozygous and pure recessive individuals of the F_1 generation should get heterozygous mates. In these few families the expectation for F_2 would then be of a 1:3 ratio for the Nn × Nn matings and a 1:1 ratio for the Nn × nn, which would obviously give one half as many affected children in the first case as in the second.

Harman's charts afford sufficient data to settle these points conclusively. In regard to the first point there are 96 cases in which the cataractous child of a cataractous parent has himself reached maturity and produced one or more normal offspring, thus proving on the assumption that cataract is recessive, that his consort was either NN or Nn. Instead of finding, as we should expect on the assumption that cataract is a recessive, that only 3 or 4 (1:30) of these individuals would find an Nn mate and therefore be capable of giving some affected children, what we really do find is that of these 96 families, 83 have produced cataractous children—86 per cent. It is significant, incidentally, that of the remaining 13, ten are families of three children or less. A more striking refutation of the assumption could hardly be found.

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[6] This statement is an estimate based on data gathered from a number of sources, *e. g.*, *Jour. Amer. Med. Ass'n*, quotations from the census reports, etc. It is probably not too low, but if the incidence were as much as 1:100 we should still find a similar but of course less marked discrepancy.

[7] This result is arrived at by the use of a formula similar to one given by Jennings in his paper "The Numerical Results of Diverse Systems of Breeding," in *Genetics*, Vol. 1, No. 1, pp. 53–89. Jennings is not responsible for the use of his formulae in this connection, but they obviously apply.

[8] Corroborative evidence on this point is furnished by the histories of retinitis pigmentosa in the paper by Usher already referred to.

In regard to the second point we find that there are 47 normal individuals in the same F_1 generation from the supposedly Nn × nn matings who have also come to maturity and produced children. In 42 of these families only normal children have resulted. In 10 per cent. of such families, however, one or more cataractous children have been produced. But the relation between 10 per cent. on the one hand and 86 per cent. on the other is very far from being the relation of one to two.[9]

These considerations based on the results from such matings as are included in category *B*, and the F_2 descendants of such matings, furnish convincing evidence that congenital cataract is not a Mendelian recessive.

Finally as to the last point, which concerns category *C*, we can agree with the authors when they say:

> The critical test as to whether or not congenital cataract can be considered as a simple recessive character lies in the matings of abnormal by abnormal. Families of this kind should have only abnormal children. Only three such matings are available.

I find two of these, but have searched the charts in vain for the third which must be an error or inadvertently drawn from outside sources. This really is immaterial since it would require many such cases to prove the hypothesis, where a single bona fide case in which two affected individuals produce normal offspring is sufficient to overthrow it. One of the three cases cited is such.

[9] How we are to explain the five individuals in this group who have produced cataractous offspring is not material to the present consideration. Not to mention the possibility of faulty classification, possible mutation etc., they might, for instance, belong to the class mentioned by Jones and Mason in which the parent while really heterozygous appears in the recessive form.

In chart 342, III–28 and III–37 are shown as a pair of parents both of whom are affected in both eyes. The descriptions quoted from Nettleship by Harman (*op. cit.,* p. 148) show that the diagnosis is evidently based on ophthalmoscopic examinations. This is a clear case of abnormal by abnormal, and if we were to regard it as "doubtful" we could find equal justification for so regarding any other chart in the whole series. The offspring of this marriage are seven children, of whom two have cataract, three thought to have been free from it died in infancy, and two are definitely known to be normal. This is the one critical case that is needed and, taken at its face value, it completely refutes the argument for the recessive nature of congenital cataract.

In conclusion, the writer does not wish to insist on arguments from a few particular cases, nor does he wish to make purely academic distinctions in the treatment of data. In particular, he does not wish to be understood as maintaining that congenital cataract behaves strictly as a single dominant unit character—a view to which he does not subscribe. The point upon which he does insist, however, is that the view, presented in the paper under discussion, namely that congenital cataract is due to a single recessive character, not only fails to find support in the data which was presented, but is in reality actually disproved by that data.

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